REMARKS

Favorable reconsideration is respectfully requested in view of the foregoing amendments and the following remarks.

I. EXAMINER INTERVIEW, CLAIM STATUS & AMENDMENTS

Applicants appreciate the telephone interview with Examiner Singh and Examiner Falk on August 20, 2007 to discuss this case.

Claims 1-3 were pending in this application when last examined and stand rejected.

Claims 1-3 have been canceled without prejudice or disclaimer thereto. Applicants reserve the right to file a continuation or divisional on any canceled subject matter.

New claims 4-8 have been added.

New claims 4 and 7 correspond to original claim 4 (elements 1(a) and 1(b), respectively). Further support can be found in the disclosure, for example, at page 11, last line, to page 12, line 10. Support for the revised method steps in claim 4 can be found in the disclosure, for example, at page 14, line 20 to page 16, line 4, and in original claim 1.

Support for new claims 5-6 and 8-9 can be found in the disclosure, for example, at page 12, lines 15-27.

No new matter has been added.

Claims 4-9 are pending upon entry of this amendment.

II. INFORMATION DISCLOSURE STATEMENT

On page 2 of the Office Action, it was indicated that JP 2002-58489 (reference AK in the IDS filed February 8, 2005) has been officially considered to the extent that it is presented in English. However, Applicants did not receive an Examiner initialed PTO-1449 form indicating such.

Applicants thank the Office for noting such and respectfully request the Office to return an Examiner-initialed PTO-1449 form (of IDS filed February 8, 2005), indicating such so the reference will be published on the face of any patent issuing from the application. Alternatively, the Office could indicate such on a Notice of References Cited.

III. ENABLEMENT REJECTION

Claims 1 and 3 were again rejected under 35 U.S.C. § 112, first paragraph, on the basis that the specification fails to enable the claimed method of screening (claim 1) and the transgenic mouse (claim 3) for the reasons set forth on pages 3-13 of the Action.

This rejection is respectfully traversed as applied to the amended claims.

With regard to claim 1, the Office, in the interview and in the Action, contends that the claim is overly broad for it encompasses any cell or any animal for screening therapeutics for complex diseases, such as diabetes and obesity. In particular, on page 6 of the Action, the Office argues that it is unclear how the skilled artisan would measure binding of ERRL1 to ERR and transcriptional activity in any type of cell.

With regard to claim 3, the Office argued that the specification does not enable using the claimed transgenic as a control in a screening method or as an animal model itself.

Applicants respectfully disagree with the Office's positions.

Nonetheless, for the sole purpose of expediting prosecution and not to acquiesce to the rejection, claims 1-3 have been canceled and replaced with new claims 4-8. It is respectfully submitted that this amendment fully addresses and overcomes the Office's concerns. Specifically, the new claims do not call for the use of any cell type for screening. Instead, the new claims call for the use of cells expressing ERRL1 and/or ERR as described in the disclosure. Accordingly, one of skill in the art would readily appreciate that not just any cell type can be used in the screening methods of the new claims. Moreover, Applicants respectfully submit that the

skilled artisan, upon reading the disclosure and in view of the knowledge in the art, could readily identify and use such cells without undue experimentation for the following reasons.

First, the Specification (at page 12, lines 15-27) describes examples of cells that express ERRL1 and ERR. These include: (1) adipocyte cells, such as established cell line 3T3-L1; (2) an established animal or bacterial cell line transfected with ERRL1 cDNA and/or ERR cDNA; and (3) isolated animal cells, such as BAT (i.e., brown adipose tissue), heart, skeletal muscle, or kidney, in which ERRL1 protein has been highly expressed. The examples in the disclosure further exemplify the use of specific cell types. It is respectfully submitted that the description and examples in the disclosure fully represent and enable the use of cells expressing ERRL1 and/or ERR as claimed. One skilled in the art could readily identify and use such cells without undue experimentation.

Second, the Specification fully describes and enables *in vitro* screening methods using such cells. See, for instance, the Specification at page 14, line 20 to page 16, line 4, which describes the *in vitro* method of the new claims, which calls for using cells to screen for a substance that increases transcriptional activity of nuclear ERR and/or promotes binding of ERRL1 to ERR. The Specification also provides *in vitro* working examples demonstrating the interaction of ERRL and ERR in a cell. See for example, Example 2.4 on pages 29-30. The results of the experiments disclosed therein demonstrate that ERRL1 can function as a protein ligand for ERRs and activate ERR-mediated transcription in cultured cells.

Third, the Specification discloses in detail the procedures and techniques required to conduct the claimed invention. Moreover, the procedures and techniques described in the Specification are common and routine in the biotech arts.

Thus, it is respectfully submitted that the description and examples in the disclosure fully represent and enable the use of cells expressing ERRL1 and/or ERR as claimed. Moreover, it is respectfully submitted the skilled artisan, upon reading the disclosure and in view of the knowledge in the art, could readily identify and use cells expressing ERRL1 and/or ERR (as

claimed) in the claimed *in vitro* screening method using the disclosed routine techniques without undue experimentation. Thus, contrary to the Office's position, the Specification is enabled for the claimed screening method using such cultured cells expressing ERRL1 and ERR.

Therefore, the enablement rejection of claims 1-3 under 35 U.S.C. § 112, first paragraph, is untenable and should be withdrawn.

IV. WRITTEN DESCRIPTION REJECTION

On pages 15-18 of the Action, claim 2 was newly rejected under 35 U.S.C. § 112, first paragraph, on the basis the Specification lacks written description support for the claimed pharmaceutical composition for the treatment of obesity and/or diabetes.

Applicants respectfully disagree with the Office's positions. Nonetheless, for the sole purpose of expediting prosecution and not to acquiesce to the rejection, the present amendment overcomes this rejection by cancelling this claim.

V. INDEFINITENESS REJECTION

Claims 1-2 were newly rejected under 35 U.S.C. § 112, second paragraph, as being incomplete for omitting essential steps for the reasons set forth on pages 18-19 of the Action.

Applicants respectfully submit that the present amendment overcomes this rejection. Specifically, the rejected claims have been canceled and replaced with new claims that fully delineate the method steps of the invention.

Thus, the 112, second paragraph, indefiniteness rejection of claims 1-2 is untenable and should be withdrawn.

V. PRIOR ART REJECTIONS

Claims 1-2 were again rejected under 35 U.S.C. 102(e) as being anticipated by Spiegelman et al. (US patent publication no. 2003/0124598; published July 3, 2003, effective filing date of November 9, 2001) for the reasons on pages 20-21 of the Action.

Claims 1-2 were rejected under 35 U.S.C. § 102(b) as being anticipated by Spiegelman et al. (WO00/32215) for the reasons on pages 22-23 of the Action.

Claim 2 was newly rejected under 35 U.S.C. § 102(b) as being anticipated by Scheen et al. (Diabetes Metab. Res. Rev., Vol. 16, No. 2, pp. 114-24, 2000) for the reasons on pages 23-26 of the Action.

Applicants respectfully submit that the present amendment overcomes these rejection as applied to the amended claims.

First, as to the rejections over Spiegelman et al. (US 2003/0124598) and Spiegelman et al. (WO00/32215), these references disclose a method for screening a substance affecting ERRL1/PGC-1beta. However, the PGC-1 of the Spiegelman et al. references is ***not synonymous*** with ERR of the present invention. The new claims clearly recite and relate to interaction between ERRL1 and ERR. As described at page 4, lines 716, the PGC-1 is a coactivator of PRARγ. The Applicants found "PGC2", which is similar to PGC-1, and a novel binding factor to PRARγ. Then, the Applicants further found that the PGC2 is a novel ligand to ERR, and re-named PGC2 to "ERRL1" (see page 4, lines 21-26 of the instant Specification).

Thus, it is clear that the PGC-1 of the Spiegelman et al. references is ***not*** the same as ERR of the present invention. Accordingly, the Spiegelman references fail to disclose or suggest each and every element of the claimed invention. For this reason, the Spiegelman references fail to anticipate the claimed invention.

Second, as to the rejection over Scheen et al., the present amendment overcomes the rejection since claim 2 has been canceled.

Therefore, the above-noted anticipation rejections of claims 1-2 over Spiegelman et al. (US 2003/0124598), Spiegelman et al. (WO 00/32215) and Scheen et al. are untenable and should be withdrawn.

VI. CONCLUSION

In view of the foregoing amendments and remarks, it is respectfully submitted that the present application is in condition for allowance and early notice to that effect is hereby requested.

If the Examiner has any comments or proposals for expediting prosecution, please contact the undersigned attorney at the telephone number below.

Respectfully submitted,

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ATTACHMENTS

1. English translation of the Abstract of JP 2002-58489 for reference AK in the IDS filed February 8, 2005.

PATENT ABSTRACTS OF JAPAN

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(54) METHOD FOR SCREENING MEDICINE THAT IMPROVES INSULIN RESISTANCE IN DIAMETES

(57)Abstract:

PROBLEM TO BE SOLVED: To immediately and fundamentally elucidate the antidiabetic mechanism of TZD, and to establish a system that serves as an index in screening a new antidiabetic medicine.

SOLUTION: This method is intended for screening a ligand to PGCW2- PPARγ complex, and comprises the steps of adding a test substance to a system consisting of (1) a fusion protein containing a DNA-binding domain and PGC2, (2) PPARγ, and (3) a vector containing a region recognized by the DNA-binding domain, a promotor, and a reporter gene linked to the promoter so as to be able to be expressed, and detecting the transcription activity regulation of PGC2-PPAγ complex using the expression of the reporter gene as an index. The present invention is also related to the fusion protein that can be used in the above method for the above screening, and to a gene encoding the fusion protein.

LEGAL STATUS

[Date of request for examination]

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